Torsion Patterns of Chordae Tendineae in Rupture of Papillary Muscle

Review of the Literature; Report of a Case

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CARDIAC RUPTURE has probably been known since the beginning of medical literature. As the study of pathological anatomy developed, however, descriptions of rupture involving only special structures of the heart appeared. Rupture of a papillary muscle was first reported by Mérat[20] in 1803. In the century that followed, however, only six additional cases were recorded, and one of the first reviews of this condition, by Voigt[28] in 1932, included only nine cases, although additional ones were then on record. The first comprehensive review of this cardiac catastrophe was prepared by Stevenson and Turner in 1935.[27] They collected reports of 19 cases and added one of their own. Reviews by other investigators[1, 3, 5, 25] since that time have established a fairly characteristic clinical picture, and the antemortem diagnosis of ruptured papillary muscle was definitely made on four occasions,[1, 3, 5, 25] and suggested on another.[1] Not all the reported cases were included in any of these reviews, however, and an exhaustive search has shown that a total of 56 examples of ruptured papillary muscle are now listed in the world medical literature.[1-3, 5-8, 10-13, 15-21, 23-29] The case here presented is the fifty-seventh. An additional instance of papillary muscle rupture terminally in the course of acute disseminated lupus erythematosus is as yet unpublished.[4]

REPORT OF A CASE

A 78-year-old Caucasian man, was admitted to the Glendale Sanitarium and Hospital at 12:25 p. m., October 2, 1954. He was apparently in shock, had cold skin, and was perspiring profusely; no peripheral pulse or blood pressure was obtainable.

A daughter-in-law, who accompanied the patient to the hospital, stated that about an hour earlier he was in the living room watching the world series baseball game on television (fourth and final game) when she suddenly was aware that the set was no longer on. She went into the room, found the television set turned off, and the patient on the floor, pulseless, with Cheyne-Stokes respiration, and unconscious. He had been having frequent anginal pains the previous week, and was known to have arteriosclerotic cardiovascular disease with coronary insufficiency and arteriosclerosis obliterans of the lower extremities.

The attending physician, who saw the patient at his home, gave him 8.0 mg. (1/8 gr.) of morphine sulfate and sent him to the hospital by ambulance. An additional 8.0 mg. of morphine sulfate and 2 cc. of levophed in saline solution were given at once. About 45 minutes later the blood pressure was barely perceptible at 45/0 mm. of mercury. Heart tones were strong and regular at 96 per minute. About this time a peculiar cyanotic mottling was noted below the level of the sixth and seventh ribs. The condition of the patient deteriorated rapidly, and about an hour and a half after admission the respiration and heart tones again became irregular. An electrocardiogram showed auricular fibrillation and lateral ischemia. Breathing became labored and the patient died at 3:05 p. m., about three and a half hours after onset of acute symptoms. No laboratory studies were completed before death.

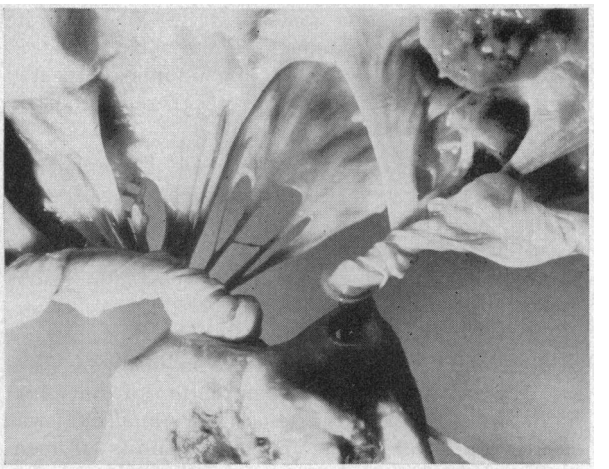

Figure 1.—Inferior surface of anterior commissure of mitral valve, showing double spiral twisting of chordae attached to necrotic avulsed papillary tip.

At autopsy the heart was observed to be enlarged, weighing 540 grams. There was left ventricular hypertrophy and dilation. The atria, the right ventricle and the valves (except the mitral) appeared normal. Cut surfaces of the myocardium showed areas of red-brown mottling in the anterior and lateral left ventricle deep to the site of attachment of the anterior papillary muscle. This muscle was ruptured in its midsection. The anterior half of the mitral valve was distorted by torsion of the chordae tendineae, which had been twisted on themselves by the tip of the anterior papillary muscle to which they were attached (Figure 1). This phenomenon was made possible by the infarction of the midsection of the papillary muscle with consequent avulsion of the tip by the mechanical trauma of cardiac contraction; this papillary tip was then apparently repeatedly thrown backward, upward, over and forward through the chordae tendineae at the anterior commissure until, after three complete revolutions, the chordae were twisted into two thick, tightly wound spirals functionally incapacitating the mitral valve so that it could neither completely open nor completely close. The lungs were moderately con-

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gested and edematous. Together they weighed 1,160 gm. Other observations were not significant.

DISCUSSION

The odd twisting of the chordae tendineae noted at autopsy in this case led to curiosity as to what mechanism might have brought it about. From a purely mechanical point of view it is apparent that two axes of rotation are possible for a fragmented tip of a papillary muscle supported by the chordae tendineae—namely, vertical rotation around the long axis of the chordae tendineae, and rotation around an axis parallel to the valve margin. In the present case the axis of rotation was the latter, and at right angles to the axis of the chordae tendineae, like the twisting arms of a gymnast "skinning the cat," or the action of a turnbuckle. Incidentally, in subsequent study of artificially produced torsion it seemed that three complete revolutions was the greatest number of turns possible. The direction of rotation was then studied and, as described in the autopsy protocol, it was seen that this rotation was backward and upward, then forward through the chordae tendineae, and down, producing the distortion as shown. Such twisting could only be produced by the "drag" of the flowing blood on the papillary fragment. After diastolic filling of the left ventricle, the direction of blood flow during systole is away from the mitral axis and upward toward the aorta, carrying the papillary fragment to a position beneath and behind the mitral valve, the end phase of systole forcing it through the chordae at the commissure into the mitral orifice, there to be caught by the diastolic inflow, and brought forward and downward to complete the revolution. The literature was reviewed to confirm or deny this concept.

Several of the early investigators noted a queer twisting of the chordae tendineae[6, 7, 16, 28, 29] but did not accurately record the direction of twisting, or attach significance to the phenomenon or attempt to explain it. Later investigators provided photographic illustrations, and in instances where these are sufficiently clear, characteristic double spiral twisting is frequently shown, identical to that seen in the specimen in the present case.[3, 14, 18, 19, 26] In other studies similar twisting was more or less clearly identifiable by description.[1 (Case 2), 3, 12, 24] In Askey's report, the photograph in Case 6 appears to show a half twist in opposite rotation, but the orientation of the photograph is not entirely clear. Vertical twisting was definitely described in only one instance (Askey, Case 1). The 56 previously reported cases and the one herein described can therefore be classified as follows:

Characteristic double spiral twisting	6
Reverse double spiral twisting (?)	1
Double spiral twisting, direction of rotation not stated	8
Vertical twisting	1
Twisting, type not identified	2
No information available	2
Twisting not present, or not mentioned	32
Incomplete papillary rupture	5
Total	57

The anatomy of the papillary muscles varies considerably, and in many cases is such that these torsion patterns cannot be produced. This undoubtedly explains the absence of twisting in so many instances.

Duration of life after rupture of a papillary muscle is usually a matter of hours, but a few cases in which the patient lived for some time are recorded. Although the interval before death is somewhat less in cases with torsion of the chordae tendineae, the difference is not significant. In the normal heart both papillary muscles have chordae tendineae connected with the respective half of each mitral cusp.[20] Therefore, in single papillary rupture there is only partial incompetence of both cusps, rather than complete functional loss of one cusp. This partial functional loss is not so severe as might be expected, owing to the support given to the loose cusp halves by the remaining intact papillary muscle and its chordae tendineae. However, when the blood flow brings about the torsion described, the chordae tendineae roll together in turnbuckle fashion, tightly drawing the cusps together, at the same time preventing complete closure. The result is mitral stenosis with fixation and incompetence, severe pulmonary congestion and edema, and systemic anoxia.

SUMMARY

A case of rupture of a papillary muscle is reported and reports of 56 cases in the world literature are reviewed. In 18 of these 57 cases (32 per cent), there was twisting of the chordae tendineae of the avulsed papillary tip. In the case here reported and in five others in the literature, the torsion pattern was a double reciprocal spiral with a characteristic direction of revolution so that the upward arc was through the commissure and the downward arc was through the central region of the mitral orifice. In ten additional instances the torsion patterns were probably identical, but not completely documented in this respect. In two other cases there were unusual torsion patterns—one around the same axis but possibly in reverse rotation, the other apparently around a vertical axis.

This apparent consistency in torsion pattern seems to provide an additional facet of interest in the study of papillary rupture. Some degree of clinical significance is also suggested, in view of the probable aggravation of the mitral deformity and dysfunction by this peculiar mechanism.

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Intercostal Arteriovenous Fistula Following Transthoracic Retroperitoneal Thoracolumbar Sympatheticoganglionectomy

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IN A BRIEF SEARCH of the literature no recent report could be found of intercostal arteriovenous fistula following transthoracic procedures or rib fractures.

CASE REPORT

The patient, a woman 45 years of age, had bilateral, two-stage transthoracic transpleural retroperitoneal sympatheticoganglionectomy involving the third thoracic through the first lumbar ganglia including the greater, lesser and least splanchnic nerves, and half of the coeliac ganglion on each side (Shumaker procedure). The kidneys and the adrenal glands were inspected bilaterally. The approach used on both sides was resection of the eighth rib and surgical fracture of the neck of the ninth rib. This procedure was done for a chronic, progressive, cerebral type of hypertension of five years' duration.

The first stage was done October 23, 1954, on the left side, and the patient recovered in the usual time of eight days. Operation on the other side was done December 1, 1954. The patient again made rapid recovery, had a decrease in blood pressure and was given neosynephrine drip for 48 hours. The pressure response was gradual. She left the hospital on the tenth day.

On the fourteenth day, pain developed in the right lateral abdominal wall over the distribution of the ninth intercostal nerve. Severe and stabbing in nature, the pain was different from that previously felt after operation. On physical examination, no additional abnormalities were noted except a continuous to and fro bruit heard over the costal-vertebral angle in the region of the fractured ninth rib.

The sound was loud and radiated superiorly for approximately 6 inches, gradually diminishing in intensity. The bruit continued with a great deal of intensity laterally over the chest almost to the sternum, where again it diminished in volume. The pulse was regular and the rate was 85, with positional variations such as are associated with postural hypotension associated with sympathectomy. The blood pressure was 140/90 mm. when the patient was supine, 100/70 mm. when sitting and varying between 80/30 mm. and 90/40 mm. when standing. At first the patient had some ringing in the ears from these changes in position, but eventually noted no bad effects and was able to be ambulatory. On the basis of the foregoing observations a diagnosis of intercostal arteriovenous fistula was made. The heart rate was somewhat accelerated following the development of the fistula, and the cardiac pulsations in the neck seemed to be more prominent.

Upon general examination it was noted that the patient was well developed and well nourished. The body weight was 180 pounds and the height

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